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OUTBREAK OF CYCLOSPORIASIS – ONTARIO, MAY 1998*

During May-June 1998, the Ontario Ministry of Health and local health departments in Ontario received reports of clusters of cases of cyclosporiasis associated with events held during May. This report describes the preliminary findings of the investigation of a cluster in Toronto, Ontario, and summarizes the findings from investigations of 12 other clusters. These investigations indicated that fresh raspberries imported from Guatemala were linked to the multicluster outbreak.

Toronto

On 2 June, Toronto Public Health was notified of a laboratory-confirmed case of cyclosporiasis in a person who attended a dinner at a hotel in Toronto on 8 May. Six other persons who attended the dinner were reported to have diarrheal illness. A case of cyclosporiasis was defined as onset of any gastrointestinal (e.g. nausea or vomiting) or constitutional (e.g. fever or fatigue) symptom 1 to 14 days after the dinner and either 1) laboratory confirmation of *Cyclospora* oocysts in a stool specimen; 2) diarrhea (i.e. three or more loose or watery stools during a 24-hour period); or 3) at least four gastrointestinal symptoms. Of the 174 persons who attended the dinner, 128 (74%) were interviewed. Of these 128 persons, 29 (23%) had illness that met the case definition; three of the 29 persons had laboratory-confirmed cyclosporiasis. The median incubation period was 8 days (range: 1 to 12 days). All 29 case-patients had diarrhea; the median duration of diarrheal illness was 7 days (range: 1 to 34 days).

Eating the berry garnish (which included raspberries, blackberries, strawberries, and possibly blueberries) for the dessert was significantly associated with risk for illness. Of the 108 persons who ate or probably ate the berry garnish, 28 (26%) became ill, compared with one (5%) of the 20 persons who did not or probably did not eat the berry garnish (relative risk [RR] = 5.2; p = 0.04, Fisher's exact test). Among the berries in the garnish, raspberries were the only berries significantly associated with risk for illness. Of the 94 persons who ate or probably ate the raspberries, 27 (29%) became ill, compared with two (6%) of the 32 persons who did not or probably did not eat the raspberries (RR = 4.6; 95% confidence interval = 1.2 to 18.3).

Other Investigations

Twelve other clusters of cases of cyclosporiasis in addition to the Toronto cluster described above have been investigated; each of the 13 clusters had two or more cases, at least one of which was laboratory confirmed. Based on preliminary data, the 13 clusters comprise 192 cases; 46 (24%) of the 192 were laboratory confirmed. The dates of the events associated with the clusters ranged from 2 May through 23 May 1998.

Fresh raspberries were the only food in common to all 13 events. Raspberries were included in mixtures of various types of berries at 12 events and were the only type of berry served at one event. The median of the event-specific attack rates for the 13 events, irrespective of exposures, was 89% (range: 23% to 100%). The median of the event-specific attack rates for persons who ate or probably ate the food items that included raspberries was 100% (range: 26% to 100%); the median attack rate for persons who did not or probably did not eat these food items was 0% (range: 0% to 67%). Eating the food items that included raspberries was significantly associated with risk for illness for five events; for the other eight events, eating the raspberry-containing food items could account for 60 (92%) of 65 cases. Traceback investigations to identify the source(s) of the raspberries have been completed for eight events, including the event described above; Guatemala was the only source of the raspberries served at the events. Mesclun

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lettuce and fresh basil, which were implicated in outbreaks of cyclosporiasis in the United States in 1997[1,2], each were served at two events but were not significantly associated with risk for illness.

Source: *Toronto Public Health, Toronto; Haliburton-Kawartha-Pine Ridge District Health Unit, Port Hope; Simcoe County District Health Unit, Barrie; York Regional Health Unit, Newmarket; Disease Control Service, Public Health Branch, Ontario Ministry of Health, Toronto; Central Public Health Laboratory, Laboratory Services Branch, Ontario Ministry of Health, Toronto; Canadian Food Inspection Agency, Fresh and Processed Plant Products Division, Ottawa, and Food Inspection, Ontario Region, Toronto and Guelph; Bureau of Infectious Diseases, Field Epidemiology Training Program, Laboratory Centre for Disease Control, and Food Directorate, Health Canada, Ottawa; Parasitic Disease Surveillance Unit, New York City Department of Health, New York; Division of Parasitic Diseases, National Center for Infectious Diseases, and an EIS Officer, Centers for Disease Control and Prevention, Atlanta.*

Editorial Note: The findings in this report indicate that fresh raspberries imported from Guatemala were linked to the outbreak of cyclosporiasis in Ontario in May 1998. Outbreaks of cyclosporiasis in North America in the spring of 1996 and 1997 also were linked to Guatemalan raspberries; the mode of contamination of the raspberries was not identified for any of these outbreaks[1,3]. No outbreaks were recognized in association with Guatemalan raspberries during Guatemala's fall and winter export seasons in 1996 and 1997.

After the outbreak in 1996, berry growers and exporters in Guatemala, in consultation with the Food and Drug Administration (FDA) and the United States Centers for Disease Control and Prevention (CDC), voluntarily introduced control measures that focused on improving water quality and sanitary conditions on individual farms[1]. In the spring of 1997, another outbreak of cyclosporiasis occurred despite the implementation of control measures and the restriction (beginning 22 April 1997) that, during that spring, only farms classified by the Guatemalans as low risk could export to North America[1]. In the spring of 1998, FDA did not allow importation of fresh raspberries from Guatemala into the United States. The Canadian Food Inspection Agency reported that fresh raspberries from farms that the Guatemalans had classified as low risk continued to be imported into Canada until 9 June 1998. The occurrence of outbreaks in 1997 and 1998 despite the implementation of control measures on Guatemalan farms suggests either that the control measures may not have been fully implemented by some farms, were not effective, or were not directed against the true source of contamination of the raspberries[1]. The Guatemalan Berry Commission and the government of Guatemala are developing a more comprehensive plan for growing and handling raspberries that includes additional control measures and inspection criteria; the plan is being reviewed by U.S. and Canadian officials.

This is at least the third, and possibly the fourth[4], consecutive year in which outbreaks of cyclosporiasis linked to consumption of raw produce have occurred in North America. In addition to Guatemalan raspberries, fresh mesclun lettuce and fresh basil that were not from Guatemala have been implicated in outbreaks in the United States[1,2]. The mode of contamination of the produce was not determined for any of the outbreaks, in part because the methods for detecting *Cyclospora* on produce and in other environmental samples are insensitive for detecting low levels of the parasite. Produce should be washed thoroughly before it is eaten; however, this practice does not eliminate the risk for transmission of *Cyclospora*[3,5,6].

Health-care providers should consider the diagnosis of *Cyclospora* infection in persons with prolonged diarrheal illness and specifically request testing of stool specimens for this parasite. The average incubation period for cyclosporiasis is 1 week; in patients who are not treated with trimethoprim-sulfamethoxazole[7], illness can be protracted, with remitting and relapsing symptoms.

Cases of *Cyclospora* infection unrelated to travel outside of Canada or the United States may be associated with a new outbreak. Newly identified clusters should be investigated to identify the vehicles of infection and to identify the sources and modes of contamination of the implicated vehicles. Although cyclosporiasis is not a reportable disease in any Canadian province or territory, as of June 1998, five states and one municipality in the United States had mandated reporting. In June 1998, the Council of State and Territorial Epidemiologists passed a resolution recommending that cyclosporiasis be made a nationally notifiable disease in the United States. In jurisdictions where formal reporting mechanisms are not yet established, clinicians and laboratorians who identify cases of cyclosporiasis unrelated to travel outside North America are encouraged to inform the appropriate local, provincial, territorial, or state health departments, which in turn are encouraged to contact, in Canada, the Division of Disease Surveillance, Bureau of Infectious Diseases, Laboratory Centre for Disease Control, telephone (613) 941-1288; and, in the United States, CDC's Division of Parasitic Diseases, National Center for Infectious Diseases, telephone (770) 488-7760.

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International Notes

ARBOVIRAL INFECTIONS OF THE CENTRAL NERVOUS SYSTEM – UNITED STATES, 1996-1997

Arboviruses include mosquitoborne and tickborne agents that persist in nature in complex cycles involving birds or mammals, including humans. Arboviral infection can cause fever, headache, meningitis, encephalitis, and sometimes death. During 1996-1997, health departments in 19 states reported to the Centers for Disease Control and Prevention (CDC) 286 confirmed or probable* cases (eight fatal) or arboviral encephalitis in humans (132 cases in 1996 and 154 provisionally in 1997). Surveillance programs in 18 states detected enzootic arboviral activity in mosquito or sentinel or wild bird populations, and cases of arboviral disease were recognized among horses or emus in 24 states. This report summarizes information about arboviral infection of the central nervous system in the United States during 1996-1997.

TABLE 1
Number and annual rate† of reported human cases of La Crosse encephalitis (LAC), eastern equine encephalomyelitis (EEE), and St. Louis encephalitis (SLE), by state – United States, 1996-1997

State	1996 population (thousands)††	LAC			EEE			SLE		
		No.		Annual rate	No.		Annual rate	No.		Annual rate
		1996	1997		1996	1997		1996	1997	
Alabama	4,273	0	0	—	1	0	0.01	0	1	0.01
California	31,878	0	0	—	0	0	—	0	1	0.002
Florida	14,400	0	0	—	1	3	0.01	0	9	0.03
Georgia	7,353	0	0	—	0	3	0.02	0	0	—
Illinois	11,847	13	3	0.07	0	0	—	0	0	—
Indiana	5,841	3	1	0.03	0	0	—	0	0	—
Kentucky	3,884	0	3	0.04	0	0	—	0	0	—
Louisiana	4,351	1	0	0.01	1	4	0.06	0	0	—
Massachusetts	6,092	0	0	—	0	1	0.01	0	0	—
Michigan	9,594	0	0	—	0	1	0.01	0	0	—
Minnesota	4,648	7	5	0.13	0	0	—	0	0	—
North Carolina	7,323	3	4	0.05	2	0	0.01	0	0	—
Ohio	11,173	20	13	0.15	0	0	—	0	0	—
South Carolina	3,699	0	0	—	0	2	0.03	0	0	—
Tennessee	5,320	1	10	0.10	0	0	—	0	0	—
Texas	19,128	1	1	0.01	0	0	—	2	0	0.01
Virginia	6,675	2	6	0.06	0	0	—	0	1	0.01
West Virginia	1,826	66	73	3.81	0	0	—	0	1	0.03
Wisconsin	5,160	8	8	0.16	0	0	—	0	0	—
Total		**125**	**127**		**5**	**14**		**2**	**13**	

† Per 100,000 population.
†† U.S. Department of Commerce, Economics and Statistics Administration, Bureau of the Census.

* For national surveillance, a confirmed case is defined as febrile illness associated with neurologic manifestations ranging from headache to aseptic meningitis or encephalitis with onset during a period when arbovirus transmission is likely to occur, plus at least one of the following criteria: 1) fourfold or greater serial change in serum antibody titre; 2) isolation of virus from or demonstration of viral antigen or genomic sequences in, tissue, blood cerebrospinal fluid, or other body fluid; or 3) demonstration of specific immunoglobulin M (IgM) antibody in serum or cerebrospinal fluid by IgM-capture enzyme immunoassay with confirmation by demonstration of IgG antibodies by another serologic assay (e.g. neutralization or hemagglutination inhibition). A probable case is defined as compatible illness occurring during a period when arbovirus transmission is likely, plus an elevated but stable (twofold or less serial change) antibody titre to an arbovirus (e.g. ≥ 320 by hemagglutination inhibition, ≥ 128 by complement fixation, ≥ 256 by immunofluorescence, ≥ 160 by neutralization, or ≥ 400 by IgM-capture enzyme immunoassay).

La Crosse Encephalitis

During 1996-1997, a total of 252 La Crosse encephalitis (LAC) cases (103 confirmed and 149 probable; one fatal) were reported from 12 states. Patients ranged in age from 5 months to 78 years (mean: 9 years), and 95% of cases occurred in persons aged < 18 years; 153 (61%) cases occurred in males, 209 (83%) in whites, and seven (3%) in persons of races other than white; in 36 (14%) cases, race was unspecified. Dates of onset of illness ranged from late June to early November. West Virginia reported 139 cases (55% of the national total), an average of 3.8 per 100,000 population per year (Table 1). Among persons aged < 18 years, who accounted for 133 (96%) of the total number of cases in West Virginia, the incidence was 15.8 per year. A fatal case occurred in a 19-month-old child in Minnesota who became ill in early August 1997 and died in November.

St. Louis Encephalitis

During 1996-1997, a total of 15 St. Louis encephalitis (SLE) cases (14 confirmed and one probable; two fatal) were reported from six states (Table 1). Patients ranged in age from 6 months to 83 years (mean: 54 years). Ten (67%) cases occurred in females; 12 (80%), in whites; and two (13%), in blacks; in one (7%) case, race was unspecified. Dates of onset of illness ranged from July 21 to late October. During 1997, Florida reported nine cases from seven central or southern counties. Enzootic SLE virus activity in sentinel chickens was detected several weeks before the first human case was diagnosed, prompting state and local public health authorities to issue public-health alerts and intensify mosquito-control measures.

Eastern Equine Encephalomyelitis

During 1996-1997, a total of 19 eastern equine encephalomyelitis (EEE) cases (all confirmed; five fatal) were reported from eight states (Table 1). Patients ranged in age from 10 months to 81 years (mean: 35 years); 10 (53%) cases occurred in males; 15 (79%), in whites; and three (16%), in blacks; in one (5%) case, race was unspecified. In all but one case, dates of onset of illness ranged from early July to mid-November. The exception was a 58-year-old man from southwestern Alabama who became ill with EEE on 8 January 1996, and died in early February. The most likely location of the patient's exposure to EEE virus was in a neighboring county at a quail farm near a hardwood swamp.

Enzootic and Epizootic Arbovirus Activity

During 1996-1997, a total of 23 states conducted surveillance for SLE, EEE, and/or western equine encephalomyelitis (WEE)

virus activity using virus isolation or antigen detection in captured mosquitoes, virus-specific antibody assays in sentinel or wild birds, or a combination of methods. Enzootic arboviral activity was reported from 18 states (Figure 1).

Figure 1. Reported enzootic activity of St. Louis encephalitis (SLE), eastern equine encephalomyelitis (EEE), or western equine encephalomyelitis (WEE) virus in mosquitoes or sentinel or wild birds, by state – United States, 1996-1997

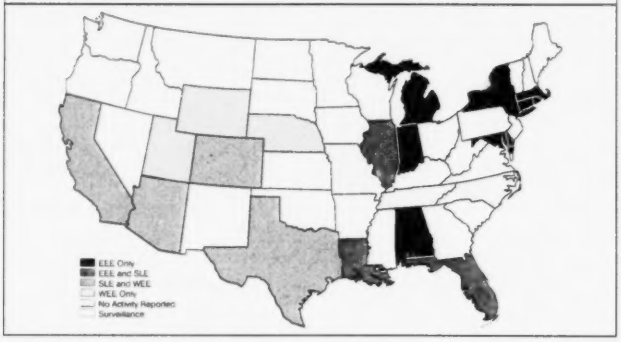

TABLE 2
Number of eastern (EEE) and western (WEE) equine encephalomyelitis cases among horses, by state – United States, 1996-1997

State	EEE		WEE	
	1996	1997	1996	1997
Alabama	9	13	0	0
Arkansas	0	1	0	0
California	0	0	1	4
Colorado	0	0	3	0
Connecticut	0	0	0	0
Florida	69	42	0	0
Georgia	5	11	0	0
Indiana	0	1	0	0
Kentucky	1	1	0	0
Louisiana	0	17	0	0
Maryland	0	0	0	0
Minnesota	2	0	1	0
Mississippi	29	4	0	0
Nebraska	0	0	1	0
New Hampshire	3	0	0	0
North Carolina	10	3	0	0
North Dakota	0	0	0	3
Ohio	0	1	0	0
Rhode Island	0	1	0	0
South Carolina	6	1	0	0
Tennessee	0	3	0	0
Texas	6	15	0	2
Virginia	5	0	0	0
Wisconsin	0	0	0	0
Total	**145**	**114**	**6**	**9**

Although arboviral disease cases among horses or other animals are not officially reported to CDC, some state health departments attempt to track such cases because cases of EEE and WEE in horses may indicate incipient human cases. During 1996-1997, a total of 274 cases of arboviral encephalitis in horses (151 cases in 1996 and 123 in 1997) were reported to public-health authorities in 21 states (Table 2). In addition, epizootics or sporadic clinical cases of hemorrhagic enterocolitis associated with infection with EEE virus (Alabama, Arkansas, Connecticut, Florida, Georgia, Louisiana, Maryland, Mississippi, North Carolina, Rhode Island, Texas, Virginia, and Wisconsin) or central nervous system disease cases associated with infection with WEE virus (California) were detected on emu farms in 14 states.

MMWR Editorial Note

During 1996-1997, LAC encephalitis remained the most frequently reported arboviral disease in the United States. The fatal case of LAC encephalitis reported from Minnesota during 1997 emphasizes that severe cases occur and can result in transient or permanent neurologic sequelae or death[2]. The incidence, public-health impact, and other aspects of this endemic disease are poorly understood and require further study.

SLE virus remains the most important cause of epidemic encephalitis in North America[3]. Surveillance for early seasonal enzootic SLE virus transmission continues to be important in detecting and controlling outbreaks and reducing human risk through vector control and modification of human activity patterns[4].

EEE is the most severe of the arboviral encephalitides, with an overall case-fatality rate of approximately 35%[5]. The fatal EEE case in an Alabama resident during 1996 was unusual because it was the first human EEE case reported from Alabama since 1965, and the onset of illness was in January. In most regions of the United States where EEE virus is enzootic, transmission to humans usually occurs during May-October[6]. This case illustrates that year-round EEE virus transmission can occur near the Gulf Coast.

The emu is an imported species of large, flightless bird farmed for meat and other products nationwide. Emus are highly susceptible to EEE virus infection, which typically results in acute hemorrhagic enterocolitis and death[7]. High-titre viremias develop in infected emus; therefore, emus may contribute to EEE virus amplification in the peridomestic environment, placing humans at increased risk.

No human cases of WEE have been reported since 1994, and only three cases have been reported during the 1990s. Reasons for the decrease in cases may include under-recognition and under-diagnosis of cases.

Health-care providers should consider arboviral infections in the differential diagnosis of all cases of aseptic meningitis and viral encephalitis, obtain appropriate specimens for laboratory testing, and promptly report cases to state health departments. Reasons for making a specific etiologic diagnosis in such cases include 1) ruling out diseases for which specific therapy is available, 2) better informing patients and their families about prognosis, and 3) alerting public-health authorities to take appropriate control measures. Human disease risk can be effectively reduced with active environmental surveillance systems and appropriate mosquito-control measures, and by providing timely information to the public.

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